

LETTERS AND
CORRESPONDENCE

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Simultaneous Occurrence of Lupus Anticoagulant and Factor VIII Inhibitors in Hemophilia

To the Editor: The letter published in the November 1997 issue, regarding simultaneous occurrence of lupus anticoagulant (LA) and factor VIII inhibitors [1,2], is very interesting. We agree with Dr. D.A. Triplett that it is of critical importance to differentiate an LA from a factor VIII inhibitor. Strict observance of the criteria recommended by the SSC Subcommittee on Lupus Anticoagulant/Phospholipid-Dependent Antibodies [3] is essential for this purpose. More than one test, with different assay principles, is necessary to screen for an LA, mainly in patients with other simultaneous coagulation defects, such as a neutralizing inhibitor or a factor deficiency. In a recently published study of 170 consecutive hemophilia A patients, we found 36 hemophiliacs who fulfilled the criteria for the diagnosis of LA, 18 of them with a time-dependent effect. It is possible that subjects with a strong time-dependent effect may have both a factor VIII inhibitor and an LA ($n = 12$) [4]. LAs were diagnosed based mainly on diluted Russell viper venom time (dRVVT). The activated partial thromboplastin time (APTT) is affected by factor VIII inhibitors; however, the platelet neutralization procedure of the APTT showed similar results to those obtained for dRVVT, except in three patients with positive dRVVT, for whom the shortening of the PNP was not sufficient to be considered positive. In our study, the Staclot®LA results agreed with those from dRVVT; 8/8 were positive on LAs and 3/3 were negative on factor VIII inhibitors. We believe that factor VIII inhibitors could be masked by LA and the misdiagnosis could delay the prescription of the appropriate therapy [5]. The above evidence stresses the need to develop a specific test to identify factor VIII inhibitors without interference of LA, and vice versa.

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Polycythemia Vera in a Patient With Acquired Immunodeficiency Syndrome

To the Editor: Hematological abnormalities are very common in patients with acquired immunodeficiency syndrome (AIDS) [1]; however, polycythemia is rarely reported in association with this disease. We report a 41-year-old man with a history of AIDS for 5 years who was found to have a high hemoglobin (Hb) on a routine CBC. He denied any shortness of breath, bleeding, or thrombotic events. His Hb had been within normal limits 6 months before. He had a history of smoking but stopped 5 years ago. His medications were Saquinavir (invirase), Stavudine (zerit), and didanosin (videx). On examination he was plethoric and had congested veins on the frontal and parietal areas of the head. His Hb was 19.6 g/dL, hematocrit 57.2%, MCV 108 fL, platelet count 167,000/ μ L, and WBC 7,100/ μ L with normal differential. Arterial blood gases showed pH 7.4, PaCO₂ 33.2 mmHg, PaO₂ 95 mmHg, HCO₃ 20.5 meq/L, and SaO₂ was 96.2%. Red cell mass was 48 mL/kg, the standard value for his height and weight is 28 mL/kg. His carboxyhemoglobin was 1.5% (normal 0–2%) and vitamin B₁₂ 1,300 pg/mL ($n = 200$ –400 pg/mL). Erythropoietin level was 7 mIU/mL (normal 0–23 mIU/mL) and leukocyte alkaline phosphatase score was 110. To the best of our knowledge there are two reported cases of polycythemia in AIDS patients in the world literature. None of them fit the criteria of polycythemia vera. Battan et al. reported a case of polycythemia in a patient with AIDS; however, this patient had an oxygen saturation of 91% [2]. Also Willocks et al. [3] reported a similar patient; however, this patient was a heavy smoker and had carboxyhemoglobin of 6.1%. Our patient fits two major criteria of diagnosis of polycythemia rubra vera (red cell mass more than 36 mL/kg and O₂ saturation more than 92% in addition to two minor criteria for diagnosis of this disease (serum B₁₂ more than 900 pg/mL and leukocyte alkaline phosphatase score more than 100) [4]. Erythrocytosis was reported once with Zidovudine (AZT) treatment [5]; however, our patient was on different medications and none of them reported this effect.

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Prothrombin Gene 20210 G-A Mutation in the Turkish Population

To the Editor: A mutation in the 3' untranslated region of the prothrombin gene at nucleotide position 20210 (G → A) was found to be associated with increased levels of prothrombin activity and with the occurrence of venous thrombosis. 20210 A allele frequency in the Dutch population was 2.3% in healthy controls and 6.2% in patients [1]. A further study in the UK population revealed almost similar results, 1.2 and 5.5% [1] respectively [2].

Turkey is situated at the meeting point of three continents of the world and stands as a crossroad between Asia and Europe. Due to its geographical location, Anatolia has historically been in contact with various races and ethnic groups from the three continents, namely Europe, Asia, and Africa. Our aim was to determine the frequency of this mutation in healthy Turkish populations from Ankara and Cyprus. With the composition of its population, Ankara represents a good example for screening studies of the Turkish population.

One hundred eighty-two apparently healthy unrelated individuals from Ankara and one hundred ten healthy individuals from Cyprus without any familial history of thrombosis, stroke, or Behçet's disease were included in the study (3).

Forty-eight patients with the diagnosis of deep vein thrombosis were also included. DNA was extracted by conventional methods and direct detection of the 20210 A allele in the prothrombin gene was performed according to a previously described method using the primers 5' TC-TAGAAACAGTTGCCTGGC 3' (nt 19889-19908) and a mutagenic primer 5' ATAGCACTGGGAGCA TTGAAGC (nt 20233-20212) Hind III (Promega, Madison, WI) to determine the mutation [1]. Genetic analysis of the factor V 1691 G-A mutation was performed as previously described [3].

The results of the distribution of PT 20210 mutation are shown in Table I. There were no individuals with a combination of FV 1691 G → A and also there were no homozygous 20210 AA individuals in all the study groups. On the other hand, during our study, we detected a healthy woman and her mother from Ankara, carrying both the variant prothrombin allele

and the Factor V 1691 (A) variant. Her maternal grandfather had PT 20210 AA in the homozygous state. None of the individuals of this family experienced a thrombotic event.

PT 20210 G → A mutation was found to be 8.1% in Turkish Cypriots, one of the highest frequencies thus far reported. A similar high frequency exist for Beta Thalassemia and FV 1691 mutation in the Turkish Cypriot population [3,4].

It is well known that co-inheritance of protein C, protein S, or anti-thrombin deficiency and FV1691 mutation confers a high risk of thrombosis [5]. However, our family data and a recent report revealed that such an effect for PT 20210 and FV 1691 was not apparent [6]. Further studies are required to resolve this question.

Our study showed that prothrombin gene 20210 G-A variation is not rare in the Turkish population and must be considered in patients with venous thrombosis.

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Elevated Beta 2-Microglobulin in Lymphorrhea From Immunoblastic Lymphoma

To the Editor: Beta 2-microglobulin (β₂m) is an 11 kDa protein recognized as a light chain component of the MHC class I antigen. The serum β₂m

TABLE I. Frequency of the Prothrombin Gene 20210 G/A Genotype in the Turkish Population

	n	Heterozygous 20210G → A	Percentage	Chromosome	PT 20210 A allele	A allele frequency
Turkish population	182	5	2.7	364	5	0.0137
Deep vein thrombosis	48	3	6.25	96	3	0.0312
Turkish Cypriots	110	9	8.1	220	9	0.0409

level is elevated in a variety of conditions characterized by lymphocyte activation and dysfunction. Recently we had the opportunity to check the $\beta 2m$ activity in the lymphorrhea of a patient with immunoblastic lymphoma. This 48-year-old patient followed for 10 years because of low-grade Non-Hodgkin's lymphoma presented with relapse localized in the right axilla as a huge mass. A biopsy revealed a transformation of the lymphoma to the high-grade immunoblastic type. The phenotype of the cells showed positivity to HLA-DR, CD20, SmIg lambda. A few days after the biopsy a collection in the right axillar area developed. A needle puncture disclosed lymph containing few lymphatic cells while the $\beta 2m$ tested by microparticle enzyme immunoassay (Abbott, Abbott Park, IL) showed a high level of 5,495 $\mu\text{g/l}$. The serum $\beta 2m$ level tested at the same time was 1,440 (normal, up to 2,000 $\mu\text{g/dl}$).

Beta 2-microglobulin is produced by nucleated cells and is detectable in the serum and other body fluids. Detection of elevated $\beta 2m$ in cerebrospinal fluid had been found as a sensitive marker for meningeal involvement in lymphoma patients [1]. Our observation shows that a localized lymphoproliferative process causes an elevation of $\beta 2m$ restricted to the involved area. Since immunoblastic lymphoma is a tumor of activated lymphoid cells, this further supports our hypothesis that $\beta 2m$ is a marker of B cell activation [2].

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Are Sickle Cell Disease Patients With Stroke Genetically Predisposed to the Event by Inheriting a Tendency to High Tumor Necrosis Factor Levels?

To the Editor: The early identification of sickle cell disease patients at risk for stroke allows transfusion therapy to be used expectantly in this group with the outcome of fewer strokes [1]. This predisposition to stroke is identified using transcranial doppler [2]. It would be advantageous if other means could be found to identify sickle cell disease patients who are at risk for stroke.

To this end we considered that a genetic predisposition to high tumor-necrosis factor- α (TNF- α) levels would serve as another helpful marker. TNF- α promotes the inflammatory process and may bring about both favorable and unfavorable effects in various target organs. Particularly high levels of TNF- α occur in patients with cerebral malaria and appear to predispose malaria patients to the occurrence of cerebral malaria [3]. The TNF2 genetic allele, in the promoter region of the TNF- α gene, is associated with these high levels of TNF- α [4]. McGuire et al. found that the TNF2 allele occurred in a significantly higher number of patients with cerebral malaria and suggested that the allele predisposed Gambian children to the occurrence of cerebral malaria [5]. Some patients with sickle cell disease have increased circulating levels of TNF- α [6]. Based on these

observations, we hypothesized that increased TNF- α plays a role in the pathogenesis of stroke in sickle cell disease, and that the TNF2 allele might be associated with increased susceptibility to stroke in these patients.

Venous blood was obtained following informed consent from sickle cell disease patients. Twenty-three had experienced at least one thrombotic stroke, and 19 had reached at least the age of 20 years without a stroke. DNA was extracted from white blood cells, tested by amplification with the polymerase chain reaction, followed by probing with allele specific oligonucleotides for the TNF1 and TNF2 alleles [5]. Three of 23 stroke patients were heterozygous for TNF1 and TNF2 alleles. The remaining 20 were homozygous for the TNF1 allele. Eight of 19 patients who had not experienced stroke were heterozygous for TNF1 and TNF2 alleles. The remaining 11 were homozygous for the TNF1 allele. The overall gene frequency for the TNF2 allele was 0.13 in the African Americans, as compared to a gene frequency of 0.16 in the Gambian population [5].

Our data do not support the hypothesis that the TNF2 allele is associated with increased susceptibility to stroke in sickle cell disease patients. In fact, the data suggest that there might be some slight protective effect of the gene against the occurrence of stroke in this population.

Since transfusion therapy is now known to be clearly beneficial [1], the need exists for additional ways to identify children with sickle cell disease who are at increased risk for stroke.

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Usefulness of IPSS for the Patients With Refractory Anemia

To the Editor: Recently an international scoring system for evaluating prognosis (IPSS) in myelodysplastic syndrome (MDS) was proposed [1] and demonstrated to be useful for predicting the outcome of patients with MDS. However, the prognosis of the patients with MDS can be predicted

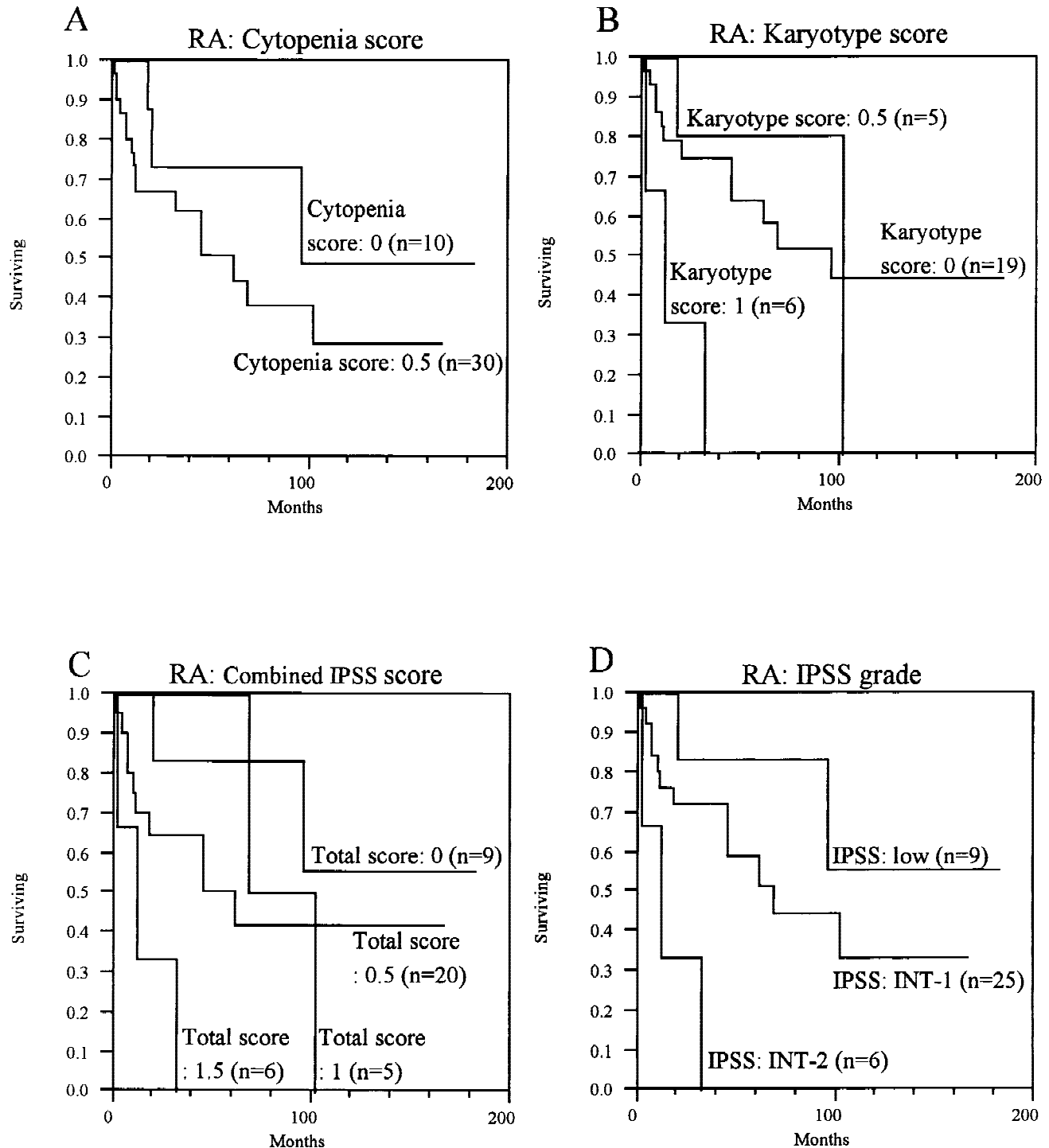


Fig. 1. Survival curves of the patients with RA according to cytopenia score by IPSS (A), karyotype score by IPSS (B), combined IPSS score (C), and IPSS grade (D).

mainly by the percentage of marrow blasts, which is almost equivalent to the FAB classification of MDS [2]. We were concerned whether the IPSS evaluation is effectively used for predicting the outcome of patients with refractory anemia (RA), whose percentage of marrow blasts is less than 5%, and is not regarded as prognostic variable in the IPSS evaluation.

Eighty patients with MDS, who were diagnosed by the criteria of the FAB proposal [2] and included 40 patients with RA, were involved in this

study. Percentage of marrow blasts, number of hematopoietic cell lineages showing cytopenia, IPSS score for chromosome abnormalities (good, intermediate, poor) [1], and IPSS grade (low, INT-1, INT-2, high) [1] were evaluated for prognostic significance. For each variable, survival curves were plotted according to the method of Kaplan and Meier. Comparison of curves was based on a Log-Rank test.

As reported previously by the IPSS group [1], each prognostic variable

was demonstrated to be closely associated with the outcomes of the patients with MDS in the present study. The patients with the stratification of low IPSS had a better prognosis compared with those with high IPSS and INT-2. The patients with IPSS INT-1 had an intermediate prognosis between low and the worst two prognostic groups (high and INT-2) (Log-Rank test: $P < 0.01$) (data not shown).

RA patients with cytopenia of none or one hematopoietic series (cytopenia score in IPSS: 0) were shown to have a better prognosis than those with cytopenia of two or three hematopoietic series (cytopenia score in IPSS: 0.5) (Fig. 1A). The patients with good or intermediate karyotype (normal and the abnormal karyotypes except for poor karyotype; karyotype score in IPSS: 0 or 0.5) had a better prognosis than those with poor karyotype (complex [≥ 3 abnormalities] or chromosome 7 anomalies; karyotype score in IPSS: 1) (Fig. 1B). The patients with an IPSS score of 0, 0.5, or 1 survived longer than those with an IPSS score of 1.5 (Fig. 1C). The stratification of the patients with RA by IPSS grade was also demonstrated to be closely associated with the prognosis (Fig. 1D) (Log-Rank test: $P < 0.01$).

In the patients with MDS, percentage of marrow blasts, biopsy features of bone marrow, NAP score, cytopenias, age, LDH value, myelodysplastic features, complication of myelofibrosis, and marrow cytogenetic patterns have been reported to be significant prognostic variables [3–5]. Among these prognostic variables, the percentage of marrow blasts that is nearly equivalent to FAB subtypes of MDS, has the most crucial prognostic significance. However, RA is defined as MDS with marrow blasts of less than 5%, which is the reason why it is difficult to predict the prognosis of RA. The present study demonstrated that the stratification of the patients with RA by IPSS grade was clearly associated with the prognosis, and the IPSS evaluation was considered to be valuable for evaluating the outcomes of the patients with RA.

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Vibrio Vulnificus Sepsis Associated With Coincidental Diagnosis of Acute Myeloid Leukemia

To the Editor: Acute myeloid leukemia (AML) may present with various infections. We present a patient in whom AML was incidentally diagnosed after sustaining septic shock due to *Vibrio Vulnificus* wound infection.

A 55-year-old woman bought a living St. Peter's fish from a fish pond in northern Israel and was wounded in her left hand while cutting its fins. Soon after, she felt pain and swelling of the hand. She was examined on the following day in the emergency room and hand surgery department. Physical examination revealed bullae and cellulitis around the puncture wound and palpable edema around the right eye but body temperature was normal. Blood for routine count, culture, and chemistry was drawn and treatment with intravenous antibiotics was started.

The patient's condition deteriorated quickly, blood pressure dropped to 90/60 and pulse was 120/min. Within a few hours the patient developed septic shock with hypotension, dyspnea, hypoxemia, anuria, jaundice, and elevated transaminase levels, necessitating treatment with fluids and vasopressors. The patient was treated intravenously with a third-generation cephalosporine and tetracycline in the assumption that *Vibrio* species might be involved in the pathogenesis. This was based on the epidemiology and the report of Bisharat and Raz [1] related to the marketing of living fish from brackish fish ponds in northern Israel. Indeed, blood cultures later grew *Vibrio Vulnificus* sensitive to the above antibiotics.

While treating this complicated patient, the laboratory informed us that the blood count was compatible with AML. Hemoglobin was 10 g/dl, white cell count was 11,000/ μ l with 80% blasts, and platelet count was 90,000/ μ l. During the following days, the patient's hemodynamic state improved but she developed necrosis of half of her palm. On the fifth day the blast count rose to 50,000/ μ l and chemotherapy with continuous infusion of ARA-C and later with Doxorubicin was started. The hand was treated in hyperbaric oxygen and only one finger had to be amputated. The patient recovered and entered a complete remission.

Vibrio Vulnificus infection may take one of three forms: wound infection with or without bacteremia, primary septicemia, and less typically acute gastroenteritis [2]. *Vibrio* septicemia is a life-threatening disease especially in patients with cirrhosis, alcoholism, and other chronic diseases, and is almost always fatal when septic shock occurs [2]. *Vibrio Vulnificus* septicemia has only rarely been associated with AML [3,4]. In our patient, the neutropenic state of the previously undiagnosed leukemia exposed her to the fulminant form of the disease. Our patient is unique in both the presentation of leukemia with *Vibrio Vulnificus* septicemia due to wound infection and in the successful treatment of both fatal diseases.

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